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This press release is an English translation of a Japanese-language press release. The official language of this press release is Japanese, and the Japanese version takes precedence over the English version in terms of content and interpretation.

<Press Release>
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Company: Chordia Therapeutics Inc
Representative: Chief Executive Officer Hiroshi Miyake
(Security Code: 190A TSE Growth Market)
Contact: Chief Financial Officer Masaru Okatani

Notice Regarding Execution of a Termination Agreement for the CTX-177 (ONO-7018) License Agreement

Chordia Therapeutics Inc (Head Office: Fujisawa City, Kanagawa Prefecture; CEO: Hiroshi Miyake, "Chordia") today announces that it has executed a termination agreement with Ono Pharmaceutical Co., Ltd. ("Ono") regarding the license agreement for CTX-177 (ONO-7018), a MALT1 inhibitor originating from Chordia's pipeline.
As disclosed in the "Notice of Discontinuation of Development of CTX-177 (ONO-7018)" dated April 28, 2025, Ono decided to discontinue the development of CTX-177 (ONO-7018) for strategic reasons and returned to Chordia the rights to develop, manufacture and commercialize CTX-177 (ONO-7018). Following this notification, Chordia and Ono have been discussing the termination of the license agreement, including the transfer of data obtained to date. Today, Chordia announces that these discussions have been completed and that the parties have executed the termination agreement.

CTX-177 is a selective inhibitor of mucosa-associated lymphoid tissue lymphoma translocation protein 1 (MALT1), a protein known to play a pivotal role in intracellular signaling in lymphoid hematopoietic cells. Since activation of MALT1 has been reported to be essential in the oncogenic transformation of lymphoid cells, inhibition of MALT1's activity by CTX-177 is expected to demonstrate antitumor effects in lymphoid hematologic cancers.

In December 2020, Ono and Chordia entered into the license agreement granting Ono the exclusive worldwide rights to develop, manufacture and commercialize CTX-177 (ONO-7018) and related compounds. Ono subsequently conducted Phase 1 clinical trials in the U.S. and Japan in patients with relapsed or refractory non-Hodgkin's lymphoma or chronic lymphocytic leukemia. As previously announced, Ono discontinued development for strategic reasons, and the license agreement has now formally concluded.
Pursuant to the terms of the license agreement, Ono will transfer to Chordia the clinical and non-clinical data obtained to date. Discussions regarding the procedures and specific conditions of the data transfer have now been finalized. The transfer will be

made without any financial consideration, and Chordia will not incur any financial burden related to the data transfer.
In addition, Chordia and Ono have agreed that Chordia will become the sole applicant for the jointly filed patent applications concerning combination therapies involving CTX-177 and other anticancer agents. Chordia will bear the associated filing and examination costs going forward.

The clinical trials conducted by Ono will be terminated under Ono's responsibility.

Chordia expects that the impact of this matter on its consolidated financial results for the fiscal year ending August 2026 will be immaterial.

Going forward, Chordia will fully leverage the clinical data obtained and consider a wide range of strategic options for the potential resumption of CTX-177 development. Despite the discontinuation by the licensee, CTX-177 continues to demonstrate strong scientific potential, and Chordia will pursue business development opportunities, including potential re-licensing, to further enhance the value of its pipeline.

About Chordia Therapeutics
Chordia's lead asset, rogocekib (CLK inhibitor CTX-712), is under Phase 1/2 clinical study in the US. Rogocekib potentially targets the vulnerability of cancer and is expected to deliver benefits to patients of various types of cancer. In addition to rogocekib, Chordia is engaged in the research and development of several assets, including CTX-177, a MALT1 inhibitor, CTX-439, a CDK12 inhibitor, and GCN2 inhibitors. For more information, please visit our website https://www.chordiatherapeutics.com/en/.